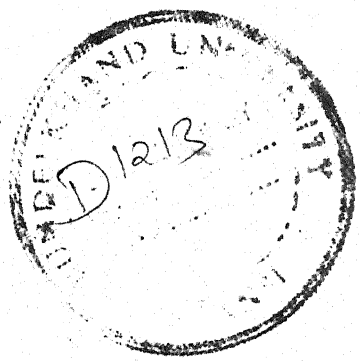
# STUDY OF ACUTE RENAL FAILURE IN BUNDELKHAND

THESIS

FOR

DOCTOR OF MEDICINE

(MEDICINE)



**BUNDELKHAND UNIVERSITY**
**JHANSI (U.P.)**

**2002** **JAI PRAKASH**

*Dedicated to respected*

*parents & daughter*

'LAKSHITA'

# *CERTIFICATE*

This is to certify that the work entitled "**ACUTE RENAL FAILURE IN BUNDELKHAND REGION**" which is being submitted as a thesis for a M.D. (Medicine) examination 2002 of Bundelkhand University has been conducted by Dr. Jai Prakash in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per University regulations.

Dated : 1/11/2001

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Dated : 1/11/2000

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Jhansi (U.P.)
(Guide)

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# ***ACKNOWLEDGMENT***

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Dated : 1/11/2001

Jai Prakash

(Jai Prakash)

# CONTENTS



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# INTRODUCTION

# ***INTRODUCTION***

Acute renal failure is characterized by an acute and usually reversible deterioration of renal functions which develops over a periods of hours to days or it can be defined as rapid decline in glomerular filtration rate over hours to days, retention of nitrogenous waste products and disturbances of extra cellular fluid and electrolyte and acid base homeostasis. The clinical features are determined by underlying condition and the rapidly developing uraemia. Many of the disorders giving rise to ARF carry a high mortality rate but if survives. Renal function usually returns to normal or near normal.

Acute renal failure complicates approximately 5% of hospital admission and upto 30% of admission to intensive care unit. Oliguria i.e. urine output less than 400ml per day is a frequent but not invariably clinical feature.

Acute renal failure is usually asymptomatic and diagnosed when routine biochemical screening of hospitalized patient reveals increased plasma urea and creatinine concentration. It may complicate a wide range of diseases, which for purpose of diagnosis and management are conveniently divided into three categories :

1) Pre renal acute renal failure (55%)

2) Renal acute renal failure (40%)

3) Post renal acute renal failure (5%)

Among these first category i.e. pre renal ARF which is due to all those diseases which cause decreased perfusion of kidney like hypovolumia due to dehydration, burns, diarrhoea, vomiting and decreased cardiac output.

Second category includes renal ARF, which arises due to diseases that involve the renal parenchyma like, acute tubular necrosis, glomerulonephritis, interstitial nephritis and renal vascular obstruction.

Third variety of ARF is post renal type which occurs because of the resistance or obstruction in the flow of urine between kidney to external urethral meatus.

It is with this background that the present study was attempted to study the incidence of acute renal failure with special reference to causative factors, laboratory investigations, complications and treatment modalities, in patients coming at M.L.B. Medical College, Jhansi.

In Bundelkhand region, which is a severely backward, poor and full of infectious disease area, the common cause responsible for acute renal failure are hypovolumia due to acute gastroenteritis, malaria and nephrolethiasis.

# AIMS AND OBJECTIVES

# ***AIMS & OBJECTIVES***

This work was designed to study the incidence and causes of acute renal failure with their treatment modalities in Bundelkhand region.

# REVIEW OF LITERATURE

# ***REVIEW OF LITERATURE***

Acute renal failure can be defined as the rapid decline in glomerular filtration rate and excretory functions occuring over hours to days leading to the retention of nitrogenous waste products in body and urinary output remains usually less than 400ml/day.

Pre-renal acute failure is present only when poor renal perfusion accounts for rapid deterioration of renal functions and the diagnosis is made by rapid return of renal functions when perfusion is restored it may occurs in depletion of the circulatory volume from blood loss, water loss, septicemic shock & low cardiac output.

GFR is auto regulated maximally at mean systolic blood pressure of 80mmHg and less than80 mm/hg is usually associated with decline in GFR.

## Classification of ARF

### *Pre Renal ARF*

i) Hypovolumia- Haemerrhage, dehydration, Burns, Diarrhoea, vomiting, over use of diuretics.

ii) Low cardiac output- Mycocardial disease, Pericardial diseases, Arrythemia, Pulm. Embolism

iii) Impaired renal autoregulation- ACE inhibitor, cyclo oxygenase inhibitor.

iv) Hyper viscosity syndrome- Polycythemia, multiple mycloma.

### *Renal ARF*

i) Glomerular disease- Glomerulonephritis, vasculitis, hemolytic uraemic syndrome, TTP, DIC, SLE.

ii) Acute tubular necrosis- Ischemia, decreased blood pressure and cardiac output, toxins,

aminoglycosides, acetaminophen, radio contrast, cisplatin.

iii) Renovascular obstruction- thrombosis, Embolism, compression.

iv) Intra tubular deposition and obstruciton- Myeloma-proteins, uric acid, oxalate, acyclovir, sulphonamide, intersticial nephritis.

***Post renal ARF***

i) ureteric- calculi, clot. Fibrosis, compression

ii) Bladder neck- BPH, Calculli, clot, cancer.

iii) Urethra- stricture, phimosis, stone.

In clinical practice pre renal ARF due to hypo-perfusion is one of the commonest cause of ARF which may accounts for 40-80% of all cases. Pre renal ARF is rapidly reversible upon restoration of renal blood flow and glomerular ultrafiltration pressure. Renal parechymal tissue is not damaged, indeed kidney from individual with pre-renal ARF function well when transplanted into recipients with

normal cardiovascular function. More severe hypoperfusion may lead to ischemic injury to renal parenchyma and lead to renal ARF.

Thus pre-renal ARF and renal ARF due to ischemia are part of a spectrum of manifestation of renal hypo perfusion. Pre renal ARF can complicate any disease that induces hypovolumia, low cardiac output, systemic vaso-dilatation & selective renal vasoconstriction.

Recent studies of patients with acute tubular necrosis emphasize that multiple results to renal functions are usually present, most common predisposing factor in the development of ATN appears to be the renal ischemia.

Coradi B et al estimated that 22% of patients over the age of 50 yrs. with advanced renal failure have ischemic renal disease.

Decrease in blood pressure due to hypovolumia leads to the activation of renin angiotensin converting system (RAAS) via cardiac baroreceptor

and carotid sinus, which causes rise in BP due to increased aldo sterone secretion and Na+ and water retention and release of ADH. Auto regularly dilatation of afferent arterioles is maximum at mean systemic arterial blood pressure of 80mmHg & hypotension below this level is associate with precipitous dicline in GFR. Lesser degree of hypotension may provoke pre-renal ARF.

Potentially reversible form of ARF can be seen when NSAIDs are given to patients with volume deplition, hypo albuminamia, and edematous disorders.

ACE inhibitor therapy may cause similar form of acute failure in patients with bilateral renal artery stenosis. So cyclooxygenase inhibitors, NSAID & ACE inhibitor are the major culprits and should be used judiciously in the setting of suspected renal hypoperfusion.

Sepsis is a major predisposing factor and nephrotoxins accounts for about 25% of all cases of

acute tubular necrosis (radio contract, aminoglycosides, heavy metals & NSAIDs).

Preponderance of ARF among elderly subjects which is probably due to the anatomical and physiological changes in the aging kidney, the incerased risk of acute renal failure related to facilitating factors- such as atheroselerosis, diabetes, hypertension, congestive heart failure and prevalence of obstructive uropathy in elderly.

Acute renal failure is an uncommon problem for the childrens. As far as the mortality is concerned, the mortality of non oliguric ARF patient to be less than the half of the oliguric ARF patients.

**Clinical presentation**

***Pre renal ARF:*** Symptom of thirst, orthostatic hypotension, tachycardias, decreased JVP, decreased skin turgor, dry month, decreased sweating, decreased urine output.

***Renal ARF:*** septic shock, H/O exposure to nephrotoxic medication or radio contrast agent, flank pain, oliguria, edema, HT.

***Post renal failure:*** suprapubic and flank pain, H/O frequency, Hasitancy, Nocturia, colicky pain radiating to groin.

Continuos renal replacement therapy & conventional hemodialysis are widely used treatment modality for severely ill patients of acute renal failure but there are yet no theraputic interventions of established efficacy in prevention of acute tubular necrosis.

Surgical interventions are best treatment modality for post renal ARF till today.

The first detailed report of fetal acute renal failure is aceredited to Hackradt a German Pathologist in 1917 was based on shouldiers who sustained severe traumatic injury.

Oliver et al (1951) distinguished iscehmic from non ischemic injury on the basis that ischemic injured cells were not confined to any one nephrone but occurred sporadically through out the nephrone & were associated with rupture of the tubular basement membrane termed as the "tubulorhexis".

Klein Knieht D et al (1972) vast majority of the cases of acute renal failure will fall into the category of pre renal form of acute renal failure or acute tubular necrosis/hemo dianamically mediated ARF.

One of the few community based studies of acute renal failure was reported from Israel by Eliahou et al (1975) this was a nation wide uremia study based on systemic screening of laboratory records of patients of age <60 yrs. for 2 yrs. periods 1965-66. Number of patients identified as having ARF was 229 with 50% greater incidence in man than womens. Age specific incidence of ARF was 10 folds more in those aged 50-60 yrs.

Dombey et al (1975) carried out a survey of biochemical results from lab. Populations of 701000, selected patients of all ages with blood urea of 100 mg/dl or greater. They identified 60 cases in intinsic ARF in a years. 1226 with pre renal type of ARF, 152 with obstructive ARF and 272 with chronic renal failure.

However prolonged pre renal azotomia can had to ischemic acute tubular necrosis with significant morbidity. Under normal circumstances renal blood flow and glomerular filtration rate are relatively constant over a wide range of renal perfusion pressure a phenomenon turned as the autoregulation. Renal autoregulation not only allows Constancy of GFR and filtered load of solutes but maintains constancy of O2 delivery, however a marked decrease in renal perfusion pressure below the auto regulatory range can load to an abrupt fall in GFR. With in autoregulatory range a fall in renal perfusion as occurs with either diminished cardiac output normally results in dilatation of glomerular

afferent arteriols and constriction of efferent arteriols. So that glomerular capillary hydrostatic pressure and GFR remain constant.

In the setting of compromised cardiac output & intravascular volume depletion, prevention of afferent artiolar dilatation (as occurs following NSAID therapy which imparts synthesis of selective eicanoids) & attenuation of efferent arteriolar constriction (as occurs following ACE inhibitor & Ca channel blocking agents) potentiates fall in GFR.

Espinel CH & Gregory AW (1980) reported that patients with pre renal azotemia had FENa less than 1% where as those with the ATN had value greater than 3%. In a subsequent study of 87 patient with ARF & plasma concentration of creatinine of over 180 μ mol/L. It was found that using FENa value of 1% divide those with pre renal failure & GN (<1%) from those with ATN & obstetrics (>1%) resulted in misclassification of one patient.

Ressmussen HH & Ibels IS (1982) defined & quantitatively assessed the risk factors and acute insults to which 143 patients with acute tubular necrosis had been exposed. 62% patients had more than one insult & 48% patients had more than one risk factors.

Myers BD et al (1982) studied 30 patients of non oliguric ARF following open heart surgery, they measured the clearance of dextran of various mole. wt. & used the result to divide the patient into those with pre renal ARF (no evidence of backleak through tubule) & ATN (evidence of back leak). The pre renal group, all of whom recovered renal functions spontaneously had average $FE_{Na+}$ of 0.5±1% where as those with evidence of back leak, 14 out of 16 of whom required dialysis, had average FENa 5.1±1.5%.

Hou et al (1983) carried out a prospective study at hospital acquired acute renal failure among 2216 consecutive medical and surgical admissions to the Tuffs, New England Medical centre in Bostan.

They found pre-renal azotemia to be the single most commmon cause of acute renal failure which accounts for about 40-80% of all cases of ARF. In this study ARF develops in 109 patients and that iatrogenic factors (drugs & sepsis) accounted for 55% of all episodes. Ten patients (1%) needed dialysis.

Moore RD (1983), demonstrated that insulin in the treatment of hyperkalemia acts by stimulating the Na-K. ATP-ase pump in skeletal and cardiac muscle, liver, fat thus driving $K^+$ into the cells. The purpose of glucose is to prevent the symptomatic hypoglycemia.

Ramsay AG et al (1983) reported a case in which by pass of an occluded renal artery produced the dramatic recovery after 47 days of oliguric acute renal failure.

Kahlmeter G & Dahlager JI (1984), In a review of 144 study of the aminoglycosides study they reported that 14% of courses of treatment with

gentamycin were associated with some evidence of this complication, with a slightly lower incidence following the use of tobramycin (12.9%), Amikacin (9.4%) & Netilmycin (8.7%).

Cleland et al (1985) states that reduction of glomerular filtration rate is usually slight & transcient on prescribing ACE inhibitors & it may even rise probably because of an improvement in cardiac output.

ACE inhibitor inhibits the conversion of angiotensin I to angiotensin II, generation of angiotensin II is fully inhibited at the low doses and at the dose used in the clinical practice have other actions such as inhibition of formation of Bradykinin from Kallikrein.

It also effects the renal circulation directly. Physiological activation of RAAS in presence of intact prostoglandin system appear to cause the vaso constriction of efferent vessels.

Kleinknicht D et al (1985) implicated the antibiotics in 136 of 398 cases with aminoglycosides responsible for 107.

Cliveland clinic have performed renal artery stents for atherosclerotic renal artery stenosis in 120 patients, the technical success was 98% the mean follow up period was 13.8 months with maximum follow up of 36.5 months. Six months patency was 95% while 12 months patency was 82.2%, BP improved in 85% of patients, renal function improved in 35% remained stable in 35% and worsened in 30% of patients.

In increased number of elderly patients with acute renal failure includes many who have wide spread atheromatous vascular disease. Occlusion caused by arterial thrombus almost invariably arises on background of long standing atheromatous renal artery disease, associated with the development of collateral blood supply from superrenal artery, lumber artery and ureteric vascular bed. In this situation the kidney remains viable after acute

occlusion of its main arterial supply even when perfusion pressure from collateral is insufficient to support glomerular filtration rate and patient is totally anuric. Renal function can reoccur after revascularization many weeks later.

Wheeler DC et al (1986) suggests that the most important prognostic factors in those with ARF is whether or not the kidney are the only organ system, that has failed. Of the other organs, the lungs are the ones that most commonly failed in conjunction with kidney. The need for artificial ventilation has a massive effect on outcome.

***Nephrotoxicity***

| | |
|---|---|
| Glomerular injury | Peneillamine, gold |
| Vascular mechanism | NSAIDS, cyclosporin , ACE inhibitor |
| Tubular toxicity | Aminoglycosides, vancomycin, amphotericin, polymixine, cisplatin, cytosinearabinosides, |

| | |
|---|---|
| | nitrosourea, 5FU, lithium, paracetamol, acyclovir, sulphonamides and methotrexate |
| Antibiotics | Penicillin, sulphonamides, cephalosporins, rifampicin |
| Antigent drugs | Allopurinol |
| Others | Phenytoin, frusemide, Ranitidine |

Smith CR et al (1986) stated the all the patients receiving aminoglycosides are susceptible to nephrotoxic complication but those with longer duration of therapy, higher plasma concentration, liver disease, advanced age, pre-existing renal impairment & (possibly) females may be at increased risk.

Gross PA & Anderson PJ (1986) in series of pediatric patients, as many as 50% of all cases of acute renal failure can be attributed to such renal parenchymal diseases as acute glomerulonephritis

and hemolytic uraemic syndrome. In hospitalized patients in whom pre renal & post renal ARF have been excluded, ARF is often due to acute tubular necrosis.

***Renal damage***

Renal damage is mainly because of acute tubular necrosis which most commonly occurs in the severely ill patients with hemodynamics disturbances or exposure to some toxic substances. This is characterized by loss of excretory function with urinary abnormality suggestive of tubular dysfunction.

Acute tubular necrosis may be aggravated by ischemia or nephrotoxins. Ischemic acute renal failure is also a part of spectrum of the menifestation of renal hypoperfusion. If differs from renal ARF where hypoperfusion induces ischemic injury to renal cells particularly tubular cells.

Basically three major categories of insults predisposing to acute tubular necrosis which

includes renal ischemia (prolonged pre renal failure). Nephrotoxins & pigmenturia (myoglobin & hemoglobin).

Course of ischemic ARF is typically classified into three phases (1) Initiation phase (2) Maintenance phase (3) Recovery phase.

Initiation phase- This phase usually lasts for hours to days and is the initial period of renal hypoperfusion during which ischemic injury is evolving. GRF declines because of :

- Glomerular ultrafiltration pressure is reduced as a consequence of fall in renal blood flow.
- Flow of glomerular filtrate within tubule is obstructed by casts comprising epithelial cells and necrotic debris derived from the ischemic tubular ephithilium.
- Back leak of glomerular filtrate through injured tubular ephithelium.

- Ischemic injury is the most prominent in the proximal tubule and thick ascending limb of loop of Henle. Cellular ischemia is triggered by-
  - Depletion of high energy nucliotides.
  - Cell swelling
  - Calcium influx & altered phopholipid metabolism.
  - Membrane injury.
  - Oxygen free radical formation.

Renal injury can be limited by the restoration of blood flow during this phase.

***Maintenance phase***

This phase lasts for 1-2 weeks during which ephithelial injury is established glomerular filtration rate stabilizes at its minimal level and urine output is lowest with the rise of uraemic

complications. Causes of low GFR despite of the correction of systemic hemodynamic includes-

- persistence of intrarenal vasoconstriction.
- Persistence ischemia of medulla triggered by release of vasoactive mediators by injured ephithilium.
- Congestion of medullary blood vessels.

Cartis & Hriek et al demonstrated the importance of angiotensin II dependent increases in efferent arteriolar tone at low renal perfusion pressure which is provided by the severe but reversible reduction in GFR which may follow prescription of ACEI to patients with bilateral renal artery stenosis or stenosis of artery to single functioning kidney.

Olin & co-workers discovered renal arteries with greater than 50% of narrowing as an incidental finding during angiography in 38% of patients with aortic aneurysm, 23% with aorto-occlusive disease & 39% with lower extremity diseases.

Jackson et al reported the incidence of acute renal dysfunctions in 38% in bilateral diseases and 80% of patients with unilateral disease precipitated by AGE inhibitors.

Recovery Phase : This is characterized by the tubular cell regeneration & gradual return of glomerular filtration rate, this phase may be complicated by marked diuresis.

Charlson ME et al (1987) prospectively measured daily S. creatinine concentration pre and post op. Cases of 278 patients undergoing elective surgery.

Mean age was 63 yrs, and many had co-morbid conditions including DM (38%), HT (76%), CVS disorder (20%). A mild rise in s.creatinine (>20%) occurred in 23% of patients the rise in s.creatinine tended to occur with in 48 hours of operation. In 12% this rise was transient & in 11% this was sustained for >48 hours.

Maillet PJ et al (1986) detected 4 out of 80 patients with acute obstruction & anuria. It should

be remembered that ultrasound detects dilatation of renal calyces & pelvis, not obstruction and the diagnosis can be missed either because of the calyces fail to dilate or do so minimally. Substantial recovery of renal function can occurs after the relief of longstanding obstruction, upto 34 days of anuria in one series of Maillet et al.

Franklin et al (1988) found that when pre renal and post renal failure were excluded, about 75% of patients in hospital with ARF had acute tubular necrosis (ATN). Clinical setting was hypotension in 90%, nephrotoxins in 19%, sepsis in 15% and rhabdomyolysis in 3%.

Turney JH et al (1988) found that the mean age of the patients with acute renal failure progressively increased from 41 years in 1950s to 61 yrs. in 1960 and they also found a striking direct correlation between mortality in ARF patients and the advancing age.

Spital A et al (1988) found that the most common cause of obstructive ARF without dilatation of renal calyces & pelvis is malignancy & he found to be diagnosis in 17 out of 25 cases.

Lens XM et al (1989), states that after giving calcium to those with life threatning hyperkalemia the plasma K+ concentration can be reduced by 1-2m mol/L over 30-60 minutes by giving iv glucose and insulin (50 ml of 50% glucose + 10 units of rapid acting insulin over 15 minutes).

Kaufman J et al (1991) found that the pre renal failure form of acute renal failure also appears to be the common cause of community acquired acute renal failures and constituted 70% of all such cases. Pre renal azotemia not only is common but also often potentially reversible.

Groenveld AB et al (1991) founds that the most common predisposing factor in the development of acute tubular necrosis appears to be renal ischemia resulting from prolonged pre renal azotemia, sepsis

& particularly septic shock has assumed an ever increasing role as a major predisposing factor in the occurance of ATN.

Prashant Adwani et al (1991) analyses the acute renal failure in western Rajasthan & study was conducted in SN Medical College, Jodhpur for period of 7 years. 163 patients of ARF were analysed retrospectively & prospectively from 91-98. Out of 163 patients of acute renal failure because of various surgical & medical causes, dialysis was done in 93 patients & 70 were treated conservatively. Common indication for dialysis were increased blood urea nitrogen (100%) & hyperkalemia (52.5%) increased mortality was found in who came with Hb <5gm% S. creatinine >4mg%. Uncounciousness presented after 48 hours & obstretic & surgical cause.

***Rhabdomyolysis***

It often occurs following traumatic muscle injury four tissue related to traumatic rhabdomyolysis are:-

- Hyperkalemia, hyperphosphatemia, hypocalcemia & metabolic acidosis occurs quickly after injury.
- Early aggressive alkaline diuresis may protect the kidney from development of failure.
- Marked positive fluid balance is often required to sustaine a brisk diuresis.
- Role of fasciotomy to prevent neuromusclar entrapment stated by Better OS & Stein JH (1990).

25% of causes of ARF occurred in the setting of nontraumatic rhabdomyolysis including strenuous exercise, seizures, heat stroke, viral infection, inflamatory myopathies, drugs like amphetamine, alcohol, HMG-CoA reductase inhibitors & metabolic disorders (hypokalemia, hyperosmolarity & hypophophatemia).

Gabow PA, Kachny WD & Kelleher SP states that the wide spectrum of rhabdomyolysis can occur following coxsackie viral infection, polymicrobial

sepsis, amoxapine overdose, extreme trauma, severe exercise & strychnine poisoning. They reported experience with 87 episodes of Rhabdomyolisis in 77 patients seen over a 4 year interval. Rhabdolyolysis was defined as increase of at least six folds in S. creatine kinase in absence of MI or CVA & an increase in M.M. isoenzyme of CK.

Cause of rhabdolyolysis included alcoholism (67%), muscle compression (39%), seizers (24%) trauma (17%), drugs (5%), metabolic (8%), muscle pain was noted in many of patients but muscle swelling was rarely present.

Feest et al (1993) carried out prospective community based study of severe acute renal failure (S. creatinine >4.5 mg/dl or 500μ mol/L).

In adults drawn from population of 44971 over a period of 2 years. Severe ARF developed in 125 adults, of whole 90(72%) were >70 yrs. of age, over all survival was 54% at 3 months & 34% at 2 yrs.

Hepato renal syndrome- this is a agrressive form of acute renal failure that frequently complicates the hepatic failure due to advanced cirrhosis or other liver disease like malignancy, billiary obstruction & hepatic recection.

Intrarenal vasoconstriction & sodium retention are the sequelae of these diseases. Portal hypertension and ascitis also have increased plasma volume but hypovolumia due to the systemic vasodilatation & pooling of blood in the portal circulation. Azotemia develops slowly over weeks or months in parallel to the deteriorating hepatic functions. In full blown hepato renal syndrome acute renal failure progress even after optimization of systemic hemodynamics, volume status & removal of nephrotoxins.

Badalamenti S et al (1993) found that the hepato renal syndrome is a life threatening complications of severe liver disease that shows many features of pre renal azotemia. In 234 patients with cirrhotic ascitis, the probability of development of this

syndrome was about 90% over a 5 year period. Severe renal vaso constriction occurs with the hepato renal syndrome and is responsible at least in large part of acute renal failure. This vaso constriction may be due to combination of enhanced renal vasoconstriction (norepinephrine, vaso pressin, endoxin, endothelins, leukotrines and increased adrenergic effects) as well as decreased vasodilators (eicosanoids, kinins and nitric acid).

Blum U et al (1993) gave local thrombolytic therapy to 14 patients with acute unilateral embolic occlusion of renal artery. In 13 patients treatment was technically successful but renal function did not recover in kidney whose artery has been completely occluded.

***Clinical Assessment***

Pre renal azotemia presents with the symptoms of thrust, orthostatic dizziness & evidence of tachycardia, decreased skin turgar, decreased

sweating, dry mucous membrane and fall in urine output.

Although ischemic and nephrotoxic acure renal failure accounts for 90% of cases of renal azotemia. Flank pain may be predominant symptom following occlusion of vessel. ARF may be associated with oliguria, edema, hypertension & evidence of hypertensive injury to other organs like LVH & LVF, retinopathy & papilloedema etc. Supra pubic and flank pain radiating to groin may suggests post renal azotemia.

D Bhowinik et al (1993) analysed the obstetric ARF in AIIMS between 93 & 99. There were 45 cases with age of 26±43 year, 15 cases were following abortion & 30 post purperial, 18 had history of hypotension, 16 due to PPH & 2 due to diarrhoea immidiately following delivery:-

| | |
|---|---|
| 27 patients- | Evidence of infection |
| 12- | Jaundice |
| 1- | HELLP syndrome |
| 7- | DIC |

| | |
|---|---|
| 11- | Retained product of conception |
| 18- | Surgical intervention was done |
| 10- | Dilatation and evacuation |
| 1- | Colpocentasis |
| 7- | Exploratory laprotomy |

15 patients dies due to septicemia, 18 recovered totally & renal biopsy done in 8. Of them 4 had cortical necrosis, 2 had microangiopathy & 2 with ATN.

***Complications***

Acute renal failure impaires the renal excretion of sodium, potassium & water & perturbs divalent cations homeostasis & urinary acidification mechanism. ARF is frequently complicated by-

Intravascular volume overload

Hyponatremia - Hypocalcemia

Hyperkalemia - Hypermagnesemia

Hyperphosphatemia - Metabolic acidosis

Anemia - Infection

Pulm. Edema anhythemia, pericarditis

GI bleeding

hyperurecemia

Hyperkalemia is most common dangerous in context of acute renal failure & is important because it can cause cardiac arrest without warning.

Freeman SJ & Fall AD (1993), states that the patient may occasionally suffer from muscular weakness or paralysis but these symptoms are rarely prominent and if they do occurs their significance is rarely appreciated (except in patients on long term dialysis).

Kaiser et al (1994) identified 105 childrens with acute renal failure (S. creatinine was greater than two folds above base line) from 21000 childrens admitted to a pediatric tertiary care centre in Philadelphia, USA over 3yrs. period. Overall

mortality of childrens was 40% with another 10% children left with permanent renal failure of the 105, 36 needed dialysis & 20 died.

Liano, F et al (1994) emphasize that multiple results to renal functions are usually present for e.g. in more than 600 patients with acute tubular necrosis, about half have more than single results to renal functions such as fever, bacteremia, endotoxemia, hypotension and aminoglycosides.

Shusterman N et al suggests that nephrotoxins accounts for about 25% of all cases of acute tubular necrosis cases. Contemporary nephrotoxins encountered includes aminoglycosides, antimicrobial agents, NSAIDS organic solvents and heavy metals like cisplatin & carboplatin.

YJ Anupama (1995), conducted a study of 80 patients in Karnataka for 4 years in 1995. Mean age was 42.75±5.26. 64 cases were due to the medical cause, 10 due to surgical cause & 6 due to obstretic cause. Acute gastroenteritis was single most

common cause for medical ARF followed by snake bite and multiorgan dysfunction. Oliguria was commonest (87.5%) presentation followed by dyspnoea 940%).

LVF seen in 31% encephalopathy in 22.5% cases, coma in 10% cases. Mean blood urea was 147.58±56.17 and mean s.creatinine was 7.08±3.43 mg%. 18 patients were treated conservatively, 62 by dialysis, 59 improved completely, 6 with partial recovery. Poor prognosis was seen in elderly surgical ARF, sepsis, multiorgan dysfunction & delayed presentation.

Frankel MC, Weinstein AM & Stanzel KH found a direct relationship between the pulse rate on initial evaluation & mortality of ARF patients and mortality of ARF patients. Mortality was 25% with pulse rate 60-69/minute &rose to 100% with PR>100/minute. A similar although inverse relationship was found with systolic BP and mortality.

T Ash et al studied acute renal failure in snake bite in M.K.C.G. Medical College, Orrisa. 21 patients of ARF following viperine snake bite were studied for 3 yrs. 16 were male and 5 were female, mean age was 30 yrs. Time interval between bite & hospital admission ws 2-80hrs. Local swelling (100%), oliguria (85%), Bleeding tendency (66%) & hypotension (33%) were initial clinical features.

Seven defects in clot quality and non clotting blood were found in 9&7 cases respectively, peak blood urea and s.creatinine levels were 147.4±7.5 & 6.5±3.6. after treatment with ASV, coagulation defects were persisted for 1-4 days but renal dysfunction for 5-20 days. 9 patients required dialysis, 4 patients dies & 2 were discharged. Renal biopsy was done in 4 patients & all showed ATN.

# MATERIAL AND METHODS

# ***MATERIAL & METHODS***

This study was carried out in the patients admitted in the wards (Medical, Surgical & Gynae/Obstretic) of M.L.B. Medical College, Hospital, Jhansi. The patients of all ages except children were included in this study. In the history following things were undertaken.

- Loose motions/vomitings present or not.
- Decreased urine output (<400ml/day).
- Abdominal pain (flank, groin region).
- Frequency of micturition, dribbling, hasitancy.
- History of accident, trauma, blood loss.
- Cardiac disease- dyspnoea, chest pain, palpitation, recurrent syneope, MI.
- History of bleeding disorder.
- H/O DM hypertension, ingestion of nephrotoxic drugs. After taking full history following clinical examination were done:

- General condition - pallor
- Pulse rate - Icterus
- Blood pressure - Cyanosis
- Hydration - Pedal edema
- Respiratory rate - Consiousness
- Temperature - abdominal exam.
- Cardiovascular exam - CNS exam.
- Resp. System exam.

After taking full history clinical examination, following investigation were performed before the start of treatment:-

Haemoglobin

Total leucocyte count

Differential leucocyte count

Erythrocyte sedimentation rate

Blood sugar fasting and 2hr Post prandial

Blood urea

Serum creatinine

Serum sodium/ serum potassium

Serum calcium/serum phosphorous/S. uric acid

Urine for albumin, sugar & microscopic examination.

Ultra sonography of abdomen for kidney size and other abdominal problem (if possible before treatment).

Electro cardigram

GFR estimation (Cockcroft gault formula)

$$\text{Creatinine clearance} = \frac{(140\text{-age}) \times \text{body wt. (Kg)} \times 0.83 \text{ (F)}}{\text{S. creatinine (mg\%)} \times 72}$$

Liver function (If required)

QBC test for malaria (if required)

***Criteria for diagnosis of ARF***

1. Urine output less than 400ml/day.
2. History <3 months.
3. Bilateral normal sized kidney (9-12cm) on ultrasonographic examination.
4. Abrupt rise of blood urea and serum creatinine.

# OBSERVATIONS

# ***OBSERVATION***

The present study was carried out in the Department of Medicine, M.L.B. Medical College, Jhansi. 50 patients of ARF were detected out of 8602 patients admitted in medical wards, surgical ward and Obst/Gynae wards in duration of one year (Oct. 2000 to Sept. 2001), with the history of decreased urine output usually less than 400ml/day. Full history was taken & clinical examination was done. Laboratory investigation were sent before the treatment was started.

## General characteristics of patients

Table I shows the distribution of patients according to age & sex. The minimum age recorded was 12 yrs. and the maximum age was 76 yrs. maximum no. of patients 22 (44%) were belongs to third and fourth decade. 31(62%) patients were male and 19(38%) were female. Male:female ratio was 16:1.

Table-I- Distribution of patients according to the age and sex.

| Age groups (Years) | Sex | | Cases | |
|---|---|---|---|---|
| | Male | Female | Total No. | % |
| <20 | 03 | 02 | 05 | 10% |
| 21-40 | 09 | 13 | 22 | 44% |
| 41-60 | 11 | 04 | 15 | 30% |
| >60 | 08 | 00 | 08 | 16% |
| Total | 31 | 19 | 50 | 100% |

Table II shows occupation wise distribution of patients. Maximum number 16(32%) were the house wives followed by farmer 10(20%). Students were 8(16%), serviceman 7(14%), Businessmen 5(10%) & labourer 4(8%) who came to this hospital.

Table-II : Distribution of patients according to the occupation

| Occupation | Cases | |
|---|---|---|
| | Number | Percentage |
| House wife | 16 | 32% |
| Student | 08 | 16% |
| Business men | 05 | 10% |
| Service | 07 | 14% |
| Farmer | 10 | 20% |
| Labourer | 04 | 08% |
| Total | 50 | 100% |

Table-III shows that total number of married male & female was 40(80%) & number of unmarried persons was 10(20%).

Table-III : Distribution of patients according to their martial status

| Martial status | Cases | |
|---|---|---|
| | Number | Percentage |
| Married | 40 | 80% |
| Unmarried | 10 | 20% |
| Total | 50 | 100% |

Table-IV : Distribution of patients according to type of acute renal failure

| Type of ARF | Cases | |
|---|---|---|
| | Number | Percentage |
| Pre-renal | 31 | 62% |
| Renal | 16 | 32% |
| Post-renal | 03 | 06% |
| Total | 50 | 100% |

Table-IV shows the different types of ARF. Patient of pre renal failure was 31(62%). In this group those patients are included who came to this

hospital with oliguria/anuria, serum creatinine <2.5mg%, Blood urea<150mg%, urine routine & microscopic examination not showing albumin, sugar, RBC, cast or pus cells & they became normal on conservative treatment. Total patients of renal type failure was 16 (32%), these patients had urinary albumin, occasional RBC & without crystals & incidence of post renal was 3(6%). All these patients had bilateral kidney size normal on ultrasonography.

Table-V : Distribution of patients according to ARF in various departments.

| Departments | Cases | |
|---|---|---|
| | Number | Percentage |
| Medical | 40 | 80% |
| Surgical | 08 | 16% |
| Obstr/gynae | 02 | 04% |
| Total | 50 | 100% |

Maximum number of patients 40 (80%) were in medical wards and minimum 2(4%) in the department

of gynae/obstr. 8(16%) patients were detected from surgical wards.

**Analysis of history & clinical examination**

Table-VI: Distribution of patients according to the cause of pre-renal ARF.

Total no. of pre renal cases= 31

| Cause of pre renal ARF (multiple responses) | Cases | | Percentage |
|---|---|---|---|
| | No. | % | Out of total cases |
| Diarrhoea/dysentry | 19 | 61.29 | 38 |
| Vomiting | 22 | 70.96 | 44 |
| Blood loss | 04 | 12.90 | 08 |
| Decreased cardiac output | 05 | 16.12 | 10 |
| Burn | 03 | 9.68 | 06 |

Table-VI shows the distribution of patients according to causes of pre-renal ARF. Total no. of cases of pre renal ARF was 31. Out of them 22(70.96) cases had vomiting followed by diarrhoea/

dysentry in 19(61.29%) cases. Blood loss was present in 4(12.90%) patients. In 5(16.12%) patients cause was fall in cardiac out put and burn was responsible for 3(9.68%) cases of pre renal ARF.

In these patients multiple response was present, many patients had more than one cause.

Table-VII – Shows distribution of patients according to cause of renal ARF

Total no. of renal ARF=16

| Cause of Renal ARF (multiple response) | Cases | | Percentage |
|---|---|---|---|
| | Number | % | Out of total case |
| Malaria | 6 | 37.50 | 12% |
| Septicemia | 4 | 25.00 | 08% |
| Cirrhosis | 2 | 12.50 | 04% |
| Aminoglycoside | 2 | 12.50 | 04% |
| Celphos poisoning | 1 | 6.25 | 02% |
| Snake bite | 1 | 6.25 | 02% |

Table-VII shows the distribution of patients according to cause of renal ARF. Malaria was commonest cause for renal ARF and was present in 6(37.50%) patients out of 16 cases of renal ARF. Septicemia was present in 4(25%) cases, 2 patients (12.50%) were found to had cirrhosis and aminoglycoside (gentamycin) toxicity. 1(6.25%) case had celphos poisoning and 1(6.25%) due to snake bite.

Table-VIII : Shows the distribution of patients according to BP (systolic) at the time of admission

| Systolic blood pressure (mmHg) | Cases | |
|---|---|---|
| | Number | Percentage |
| Not recordable | 3 | 6% |
| <60 | 1 | 2% |
| 60-79 | 8 | 16% |
| 80-99 | 17 | 34% |
| 100-120 | 11 | 22% |
| >120 | 10 | 20% |

Table-VIII shows that maximum number of patients 17(34%) had systolic blood pressure in the range of 80-99mmHg. In 3(6%) patients blood pressure was not recordable at all, these were those patients who came to this hospital with marked dehydration or haemorrhage & with low general condition. 10 patients had normal blood pressure above 120mmHg, 11(22%) patients fall in the range of blood pressure of 100-120 mmHg & 8(16%) in range of 60-79mmHg.

## Analysis of laboratory investigations

Table-IX : Shows the distribution of patients according to blood urea levels. (n=50)

| Blood urea level (mg%) | cases | | Case fatality | |
|---|---|---|---|---|
| | No. | % | No. | CFR |
| 50-100 | 21 | 42 | 5 | 23.8 |
| 101-150 | 18 | 36 | 6 | 33.3 |
| 151-200 | 06 | 12 | 2 | 33.3 |
| >200 | 05 | 10 | 2 | 40.0 |
| **Total** | **50** | **100** | **15** | **30.0** |

Maximum number of patients 21(42%) were found to have blood urea level in the range of 50-100mg%. Out of 21(42%), 5 patients (23.8%) died. Blood urea level 101-150mg% was present in 18(36%) patients of which 6(33.3%) died. 6(12%) patients had urea level in range of 151-200mg%. Of them 2(33.3%) died & also 2(33.3%) patients died out of 5(10%) patients having blood urea level>200mg%. Total cases fatality was 15(30%) out of 50 cases studied. Case fatality was gradually increasing with the rise of blood urea level.

Table-X : Shows the serum creatinine levels (n=50)

| S. creatinine level (mg%) | Cases | | Case fatality | |
|---|---|---|---|---|
| | No. | % | No. | CFR |
| 1.5-2.5 | 28 | 56 | 7 | 25 |
| 2.6-3.5 | 06 | 12 | 2 | 33.3 |
| 3.6-4.5 | 09 | 18 | 4 | 44.4 |
| >4.5 | 07 | 14 | 2 | 28.6 |
| **Total** | **50** | **100** | **15** | **30.0** |

Table-X : shows serum creatinine level 1.5-2.5mg% was present in maximum number of patients 28(56%). Out of these 28, 7(25%) died. 4(44.4%) patients died out of 9(18%) who had serum creatinine level in range of 3.6-4.5mg% & 2 cases expired out of 6(12%) & 7(14%) patients having serum creatinine level 2.5-3.5mg% & >4.5mg% respectively.

Table-XI : Shows the distribution of patients according to serum sodium level . (n=50)

| Serum sodium level (meq/L) | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| <110 | 5 | 10 | 2 | 40 |
| 111-120 | 8 | 16 | 3 | 37.5 |
| 121-130 | 12 | 24 | 3 | 25 |
| 131-140 | 18 | 36 | 3 | 16.6 |
| >140 | 7 | 14 | 2 | 28.6 |
| **Total** | **50** | **100** | **13** | **26.0** |

Table-XI shows that majority of patients 18(36%) were having sodium level in range of 131-140meq/L, of which 3(16.6%) patients died. 3(37.5%) died out of 8(16%) with the serum sodium level of 111-120meq/L. Only 2(40%) patient died out of 5(10%) & patients having sodium level of <110meq/L. 3(25%) patients died out of 12(24%) cases who had serum sodium level in the range of 121-130meq/L and 2(28.6%) died out of 7(14%) cases having serum sodium level >140meq/L.

There was no significant relationship between fatality & serum sodium levels.

Table XII shows that the case fatality was related to the level of hyperkalemia. 5(41.7%) patients expired out of 12(24%) patients having the serum potassium level in the range of 5.1-6.0meq/L.

There was no fatality in 7(14%) patients with the serum potassium levels 3.6-4.0 meq/L. 41-45meq/L serum potassium was present in 8(16%) cases, of which half died. 2 patients died out of 9(18%) & 8(16%) pateints who had serum potassium level 3.0-3.5meq/L & 4.6-5.0meq/L respectively. 2(33.3%)

patient died out of 6(12%) with severe hyperkalemia i.e S.potassium level >6.0meq/L. this shows that case fatality was directly proportional to the hyperkalemia.

Table-XII : Shows serum potassium levels (n=50)

| Serum potassium (meq/L) | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| 3.0-3.5 | 09 | 18 | 2 | 22.2 |
| 3.6-4.0 | 07 | 14 | 0 | 0 |
| 4.1-4.5 | 08 | 16 | 4 | 50 |
| 4.6-5.0 | 08 | 16 | 2 | 25 |
| 5.1-6.0 | 12 | 24 | 5 | 41.7 |
| >6.0 | 06 | 12 | 2 | 33.3 |
| Total | 50 | 100 | 15 | 30.0 |

Normal values of $S.K^{+}$ =3.5-4.5meq/L

Table XIII : Shows the serum calcium levels along with case fatality

Normal value-9-11mg% (n=50)

| Serum calcium (mg%) | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| <8.0 | 7 | 14 | 0 | 00.00 |
| 8.1-9.0 | 13 | 26 | 6 | 46.12 |
| 9.1-10.0 | 15 | 30 | 3 | 20.00 |
| >10.0 | 15 | 30 | 5 | 33.33 |
| Total | 50 | 100 | 14 | 28.00 |

Table XIII shows that 7(14%) patients with serum calcium level less then 8.0mg% had no fatality. 6(46.12%) patient died out of 13(26%) patients having serum calcium level in the range of 8.1-9.0mg%. serum calcium ranges from 9.1-10.0mg% in 15(30%) patients, of which 3(20%) patients expired & 5(33.3%) cases died out of 15(30%) patients with the serum calcium level >10mg%.

This shows that the mortality had direct relationship with hyporcalcemia.

Table XIV: shows the serum phosphates levels.

Normal vale=2.5-4.5mg% (n=50)

| Serum phosphate (mg%) | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| 2.5-3.5 | 21 | 42 | 5 | 23.8 |
| 3.6-4.5 | 12 | 24 | 5 | 41.7 |
| 4.6-5.5 | 12 | 24 | 4 | 33.3 |
| >5.5 | 5 | 10 | 1 | 20.0 |
| Total | 50 | 100 | 15 | 30.0 |

This table shows the serum phosphate levels. Maximum number of patients 21(42%) falls in the range of normal serum phosphate levels i.e. 2.5-3.5%. out of them 5(23.8%) died. In range of s.phosphate level 3.6-4.5mg%, 12(24%) patients were found & 5(41.7%) of them expired. 12 (24%) patients also had s.phosphate level 4.6-5.5mg% & out of them 4(33.3%) died. Only 5(10%) patient had

serum phosphate level more then 5.5mg% with one fatality.

Table-XV shows serum uric acid levels.

Normal value- 2.5-7.0mg% (n=50)

| Serum uric acid (mg%) | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| 2.1-3.0 | 1 | 2 | 0 | 00.0 |
| 3.1-4.0 | 7 | 14 | 1 | 14.3 |
| 4.1-5.0 | 5 | 10 | 2 | 40.0 |
| 5.1-6.0 | 17 | 34 | 4 | 23.5 |
| 6.1-7.0 | 18 | 36 | 8 | 44.4 |
| >7.0 | 2 | 04 | 0 | 00.0 |
| Total | 50 | 100 | 15 | 30.0 |

This table shows that there almost all patients included in this study were have normal uric acid level. 18(36%) patients were observed to have serum uric acid level in range of 6.1-7.0mg%, out of them 8(44.4%) were died. 4(23.5%) patients were died out

of 17(34%) who had s.uric acid level in range of 5.1-6.0mg%. only 1(14.3%) patient was died out of 7(14%). Only one case was having s.uric acid level less than 3.0mg% & was alright. 2(40%) patient died out of 5(10%) cases, having s. uric acid in range of 4.1-5.0mg%.

Table-XVI : Shows the routine & microscopic findings in urine. (n=50)

| Urine findings (multiple response) | Cases | |
|---|---|---|
| | Number | Percentage |
| Nil | 31 | 62% |
| Albumin (+,++) | 14 | 28% |
| RBCs (>5.0) | 3 | 06% |
| Pus cells | 8 | 16% |
| Crystals | 1 | 02% |
| Sugar | 1 | 02% |

**This table XVI shows the distribution of patients according to the finding of urine routine & microscopic examination.**

Urinary findings was totally nil in 31(62%) patients out of 50 patients. Albumin was present in 14(28%) patients. 3 patients had RBCs in their urine examination & two patients had oxalate crystals & sugar.

All those patients who had no abnormal finding on urine examination were ARF of pre renal type.

## Analysis of treatment & Results

Table-XVII : Shows the treatment modality used along with case fatality. (n=50)

| Treatment modality | Cases | | Cases fatality | |
|---|---|---|---|---|
| | Number | % | Number | CFR |
| Dialysis | 06 | 12 | 02 | 33.4 |
| Conservative | 41 | 82 | 13 | 31.5 |
| Surgery | 03 | 06 | 00 | 00.0 |
| Total | 50 | 100 | 15 | 30.0 |

Table XVII shows the treatment modality used with fatality. About 41(82%) patients were treated conservatively (iv fluids, antimalarial drugs,

antibiotics, blood transfusion, as per the cause) out of which 13(31.5%) patients died. Most of the patients of pre renal ARF belong to this group. Dialysis was done in only 6(12%) patients, 2(33.4%) of them died. Surgery was advised in 3(6%) patients, none of them died.

Table-XVIII : Shows the results associated with morbidity.

| Results | Cases | |
|---|---|---|
| | Number | Percentage |
| Expired | 15 | 30% |
| Complete recovery | 22 | 44% |
| Partial recovery | 8 | 16% |
| Absconded | 2 | 4% |
| LAMA | 3 | 6% |
| Total | 50 | 100% |

Table-XVIII shows that 15(30%) patients died out of 50 cases. 22(44%) discharged after complete recovery, 8(16%) discharged after partial recovery, 2(4%) absconded, 3(6%) cases leave the hospital against medical advise.

## Ultra Sonography

All these patients, studied had bilateral normal size kidneys.

# DISCUSSION

# ***DISCUSSION***

The present study was carried out on 8602 patients admitted in the medical wards, surgical wards and Gynae & Obstr. Wards of M.L.B. Medical College, Jhansi in last one year (Oct. 2000- Sep. 2001). About 50 patients were detected as having acute renal failure who constituted the material of the present study.

Out of these 50 patients, 31(62%) were male and 19(38%) were female. Maximum number of patients 22(44%) were from young age group i.e. 21-40 yrs. 15(30%) were middle aged i.e. 41-60 yrs, 8(16%) were above 60 yrs and they all were male. 40(80%) patients out of 50 patients were married & remaining 10(20%) were unmarried. Occupation wise maximum number of patients 16(32%) were housewives, 10(20%), farmers, 8(16%), students, 7(14%), service man, 5(10%) were businessmen & 4(8%) were labourer.

Eliahau et al (1975) reported that common age for the acute renal failure was 50-60 years. It was more common in males as compared to females. In the present study reason for involvement of young age may be poor hygienic condition, low literacy and poor economic status of this region.

Regarding incidence of different types of acute renal failure, pre renal failure was maximum i.e.31(62%), renal type of failure was 16(32%) and post renal failure was 3(6%). Kaufman J. et al (1991) found the pre renal type of acute renal failure constituted 70% of all cases and he has also reported that pre renal azotamia not only is common but also potentially reversible. Dombay et al (1975) studied 701000 population and they reported that pre renal cases were 1226 (0.017%), renal ARF 60(0.008%) and post renal failure ARF was 152 (0.02%). Which had very much difference as compared to this study.

Hou et al (1983) also reported that pre renal azotemia to be a single common cause of ARF which

accounts for 40-80%. Klein Knieht D. et al (1972) states that vast majority of ARF will fall into the category of pre renal form.

Maximum number of patients due to medical ARF was 40(80%) followed by surgical ARF by 8(16%) then obstretical ARF i.e. 2(4%). Y.J. Anupama (1995) reported in a study of Karnataka that out of 80 patients; 64(80%) were due to medical cause & 10(12.5%) due to surgical cause and 6(7.5%) due to obstretic ARF and acute gastrocuteritis was the most common cause for medical ARF.

The most important clinical presentation was oliguria in 31(62%), vomiting in 22(70.96%) patients followed by loose motion in 19(61.29%) patients, blood loss in 4(12.9%), decreased cardiac output in 5(16.12%) and burn in 3(9.68%) cases. It shows that acute gastrocuteritis (AGE) was the commonest (82%) cause of acute renal failure. Y.J. Anupama (1995) reported also that the acute gestro enteritis was single common cause for medical ARF & oliguria was common in 87.5%. Anderson et al

1977 reported as many as 50% of cases are not oliguric. Maillet P.J. et al (1986) detected 4 out of 80 patient without obstructive anuria.

Malaria was the commonest factor in the renal type of acute renal failure and was present in 6 patients (37.50%) followed by septicemia in 4(25%), cirrhosis in 2(12.50%), aminoglycosides (gentamycin) toxicity in 2(12.50%), celphos poisoning and snake bite was present in 1(6.25%) cases each. Kahlmeter G & Dahlagar JI (1984) reported that gentamycin (14%) was associated with same incidence of aminolgycoside toxicity.

Prevalence of malaria is very common in Bundelkhand region because this area is very poor hygienically and economically.

At the time of admission blood pressure was not recordable in 3(6%) patients who came to hospital in shock and the remaining patients blood pressure was with in normal range.

About 21(42%) out of 50 patients had high blood urea levels in the range of 50-100mg% and 18(36%) case had moderate rise in urea which was in range of 100-150mg%. Y.J. Anupama (1995) found the blood urea level 147.58±56.17 in study of 80 patients of acute renal failure.

Serum creatinine is an important marker to assess the severity of renal failure. 56% patients had serum creatinine in the range of 1.5-2.5 mg% and these patients had mainly pre renal type of acute renal failure and became normal on correction of dehydration and volume deficits. 7(14%) cases had rise in s.creatinine level >4.5mg%. 2(28.6%) of them died. The cause of death was septicemia and acute renal failure. Charlson M.E. et al (1987) found mild rise in S.creatinine in 23% of patients.

Amongst the complications of acute renal failure, Hypocalcemia, hyponatremia, hyperuricemia, hyperkalemia and metabolic acidosis were detected.

Hyponatremia (s.Na+ <130meq/L) was present in 25 (50%) patients. Of them hyponatremia was severe i.e. S. $Na^+$ was<110meq/L in 5(10%) cases and 2(40%) of them died.

Hyperkalemia ($S.K^+$ >4.5meq/L) was observed in 26 cases (52%) out of 50 cases. 6(12%) case had severe hyperkalemia ($S.K^+$ >6.0meq/L) and 3(50%) of them died and remaining patients were sent for dialysis. It shows that fatality had direct correlation with hyperkalemia.

Some of these patients were having complaint of muscle ache and weakness also. 20(40%) cases had $S.K^+$ level in range of 4.5-6.0 meq/L. They were treated conservatively i.e. 50ml of 50% glucose and 10 unit of rapid acting insulin over 15 minute. Lens X. M. et al (1989) states that the plasma $K^+$ concentration can be reduced by this method.

Hypocalcemia (s.Ca++ <9mg%) was present in 20 patients out of 50. Of them S. $Ca^{++}$ level was 8.1-9.0mg% in only 13 (26%) patients and $S.Ca^{++}$ was

<8.0mg% in 7(14%). No fatality found in patients having S.$Ca^{++}$ level <8.0mg%, these patients were treated conservatively.

Hyperphosphatemia (>4.5mg%) was observed in 17(34%) cases, with the death of 5 patients (29.4%). Remaining cases had s. phosphate with in normal values. In 2(4%) patients hyperuricemia was detected without mortality and treated conservatively.

On urine routine and microscopic examination most of the patients 31(62%) had no evidence of albumin, RBCs, pus cells or crystals, these were mainly those patients who had pre renal type of acute renal failure. These patients had multiple responses. In 14(28%) cases albumin was present showing the glomerular involvement. 3 patients (6%) out of 50 had RBCs in their urine and one patient had crystals.

On ultrasonographic examinations of abdomin, all these patients had normal sized kidney (9-12cm).

Out of 50 patient studied 41(82%) patients were treated conservatively with IV fluids, blood transfusion, antibiotics and symptomatic treatment. 2 patients (33.4%) died out of 6(12%), who were advised for dialysis and 3(6%) were advised for surgical intervention as they had post renal type of ARF due to bladder neck obstruction, urethral calculas and BPH. Prashant Advani et al (1991) suggests that out of 163 patients, 93 patients (57%) were gone for dialysis and 70 patients (43%) were treated conservatively. This difference is because of more incidence of acute renal failure in Bundelkhand region. Common indication for dialysis were increased blood urea (>100mg%) and hyperkalemia (>6.0meq/L). T Ash et al found that out of 21 patients of ARF with snake bite, 9 patients (43%) were dialysed and 4(44%) died. Y.J. Anupama (1995) stated that out of 80 patients 18(22.5%) were treated conservatively and 62(71.5%) were dialysed. Kaiser et al (1994) found that out of 105 cases 36(34%) needed dialysis and 20 cases expired.

Regarding results the 15(30%) patients out of 50 cases died. 22(44%) were discharged after complete recovery and 8(16%) discharged after partial recovery. 2(4%) patients absconded from hospital and 3(6%) cases were gone against medical advice.

D. Bhowinik et al (1993) found that out of 45 cases of obstetrical renal failure. 15(33.3%) were expired and 18(40%) were recovered completely. Poor prognosis was observed in elderly patients, surgical ARF, septicaemia, multiorgan dysfunction and delayed presentation. Y.J. Anupama (1995) suggests that 59(73%) case out of 80 were improved completely and discharged, as these patients had pre renal failure as compared to renal or post renal failure.

# CONCLUSIONS

# ***CONCLUSIONS***

In this study 50 patients were detected as having acute renal failure out of 8602 patients admitted in medical, surgical and obs/gynae wards of M.L.B. Medical College, Jhansi, in the duration of last one year from Oct. 2000 to Sept. 2001.

The following conclusions were drawn from this study:-

1. Incidence of acute renal failure was 0.58%.
2. Acute renal failure was more common in males and maximum no. of patients (44%) were from younger age groups (21-40 yrs).
3. Incidence of acute renal failure was more in house wives as compare to other social groups.
4. 80% patients were detected due to medical ARF, 16% due to surgical ARF and 4% due to obstetrical ARF.

5. Incidence of pre renal type of ARF was maximum about 62%, renal ARF was 32% and post renal ARF was 6%.

6. Acute gastroenteritis was most common cause for medical ARF.

7. Common presentation of patient were oliguria in 31(62%) cases, vomiting in 22(70.96%), loose motion in 19(61.29%) cases, blood loss in 4(12.9%) and decreased cardiac output in 5(16.12%) cases.

8. Malaria was detected as a causative factor for renal type of ARF which was in 6(37.50%) cases followed by septicemia in 4(25%) cases.

9. All patients of pre renal failure had blood urea level around or less than 100mg%. 21(42%) cases had blood urea levels in range of 50-100mg%, 18(36%) had blood urea level 100-150mg%, 6(12%) had blood urea level 150-200mg% & 5(10%) cases had blood urea level >200mg%.

10. Serum creatinine was important marker to assess the severity of renal failure. 56% patients had s. creatinine in the range of 1.5-2.5mg%, 14% patients had s.creatinine level >4.5mg% (renal type).

11. Hyperkalemia was observed in 26(52%) cases, hyponatremia in 25 (50%) cases, hypocalcemia in 20 (40%), hyperphosphatemia in 17 (34%), hyperuricemia in 2(4%). Hyperkalemia was the commonest complication of ARF.

12. On routine and microscopic examination of urine, 31(62%) patients had no abnormal finding. 14 (28%) shows albumin, 3(6%) had RBCs, 8(16%) had pus cells and 1(2%) with crystals.

13. 41(82%) patients were treated conservatively, dialysis was advised in 6(12%) patients and surgery was done in 3(6%) patients.

14. On ultra sonography examination, all these cases had bilateral normal sized kidney.

15. Out of 50 patients 15 (30%) died. 22(44%) were dischraged after complete recovery and 8(16%) discharged after partial recovery. 2(4%) patients absconded and 3(6%) leave the hospital against medical advice.

16. Fatality was related to elderly patients, hyperkalemia, septicemia, those who developed ATN and other associated illnesses like cerebral malaria, severe electrolytic disturbances etc.

Bundelkhand region has very wide range of population from rural areas. The percentage of patients attending to this hospital is less because other hospitals and health centres of Bundelkhand region had wide coverage of patients. As this study is hospital based so actual figure of incidence of acute renal failure in Bundelkhand region can not be detected, only quantum of disease i.e. ARF in patients attending to this hospital can be estimated. This was the limitation of this study

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# MASTER CHART

| S.No. | Name | Age/Sex | Marrital Status | Occupation | BP (mm/Hg) | Oliguria <400ml/day) | Bl. Urea (mg%) | S. creat. (mg%) | S. $Na^+$ (Meq/L) | S.$K^+$ | S. $Ca^{++}$ (mg%) | S.$PO^{4-}$ (mg%) | S. uric acid (mg%) | Kidney size | Cause of ARF | Type of ARF | Urine [ R & M] | Hb% | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | Nathuram | 65 yrs/M | M | Farmer | 86/50 | + | 85.00 | 3.80 | 126.0 | 6.2 | 10.50 | 3.5 | 6.7 | N | Malaria | B | alb +, pus cell + | 8.5 | E |
| 2. | Nandram | 60 yrs/M | M | Laborour | 90/60 | + | 207.00 | 1.90 | 136.0 | 3.8 | 9.50 | 3.0 | 6.0 | N | AGE | A | Nil | 10.7 | D1 |
| 3. | Ramgopal | 40 yrs/M | M | Farmer | 82/40 | + | 84.00 | 1.60 | 124.0 | 3.3 | 10.00 | 3.2 | 6.5 | N | AGE | A | Nil | 7.5 | D1 |
| 4. | Maniram | 55 yrs/M | M | Mason | 90/62 | + | 156.00 | 18.60 | 116.7 | 3.38 | 8.80 | 5.0 | 6.2 | N | Infection | B | alb++, Acc RBC | 8.7 | E |
| 5. | Radhelal | 50 yrs/M | M | Farmer | 82/60 | + | 142.00 | 7.86 | 120.0 | 3.5 | 9.40 | 4.5 | 6.0 | N | Infection | B | alb+, Acc pus cell | 8.9 | F |
| 6. | Ramwati | 22 yrs/F | U | House worker | 60/40 | + | 109.30 | 1.70 | 124.9 | 3.75 | 9.08 | 3.2 | 5.5 | N | AGE with shock | A | Nil | 12.5 | D2 |
| 7. | Shambhu Dayal | 66 yrs/M | M | Service | NR | + | 208.00 | 5.46 | 121.5 | 4.12 | 8.75 | 5.0 | 8.40 | N | Infection | B | Alb traces, Acc pus cells | 16.2 | E |
| 8. | Shobaram Mishra | 40 yrs/M | M | Farmer | 98/60 | Anuria | 223.00 | 17.68 | 145.0 | 3.28 | 9.50 | 3.0 | 6.40 | N | Malaria | B | alb+ | 8.8 | D1 |
| 9. | Kailash Narayan | 45 yrs/M | M | Business | 136/90 | + | 68.82 | 4.33 | 128.0 | 3.2 | 7.50 | 4.0 | 6.5 | N | Urethral calculus | C | alb+ ptenty pus cell sugeon, nil | 16.9 | D1 |
| 10. | Laxmi Prasad | 45 yrs/M | M | Farmer | 130/90 | + | 87.00 | 2.70 | 115.0 | 2.5 | -- | 3.5 | 5.8 | N | Cirrhosis with ascitis | B | alb+, Acc pus cells | 11.9 | D2 |
| 11. | Sukhu | 47 yrs/M | M | Mason | 110/68 | + | 86.00 | 6.90 | 140.7 | 4.5 | 6.80 | 3.0 | 5.8 | N | Urinary tract infection | B | alb+, 15-20 pus cells | 8.5 | D1 |
| 12. | Mansharam | 62 yrs/M | M | Farmer | 102/60 | + | 165.00 | 2.76 | 130.0 | 3.6 | 8.80 | 3.2 | 6.0 | N | Malaria | B | Nil | 9.5 | D1 |
| 13. | Anita | 22 yrs/F | U | House wife | 90/62 | + | 226.00 | 3.82 | 142.0 | 4.4 | 10.62 | 4.0 | 6.34 | N | Snake bite | B | Nil | 13.5 | E |
| 14. | Lad Kunwar | 42 yrsF | M | House wife | 148/90 | + | 155.00 | 8.80 | 152.0 | 3.8 | 11.50 | 4.2 | 5.62 | | Bladder neck obstruction | C | oxal ah++ Acc RBC + | 12.5 | D1 |
| 15. | Raj Kumar | 28 yrs/M | M | Student | 82/60 | + | 102.00 | 2.20 | 112.0 | 2.8 | 6.60 | 3.0 | 5.42 | N | AGE | A | Nil | 15.6 | D1 |
| 16. | Sheela | 45 yrs/F | M | House wife | 70(SBP) | ++ | 80.00 | 1.90 | 110.0 | 6.2 | 7.50 | 5.0 | 3.20 | E | AGE | A | Nil | 8.6 | D1 |
| 17. | Meena | 35 yrs/F | M | House wife | 126/80 | + | 213.00 | 9.00 | 128.0 | 5.8 | 7.80 | 4.62 | 6.20 | N | Malaria | B | Nil | 8.2 | D1 |
| 18. | Rani | 26 yrs/F | U | Student | 90/60 | - | 60.00 | 2.40 | 138.0 | 5.3 | 6.20 | 3.60 | 6.40 | N | AGE with pregnancy | A | alb+, nil | 8.4 | D2 |
| 19. | Radhe Shyam | 48 yrs/M | M | Service | 146/90 | + | 132.00 | 2.60 | 128.0 | 3.6 | 8.80 | 2.80 | 5.40 | N | Drug induced | B | alb++, surgery + RBC 20=26 | 10.6 | G |
| 20. | Rajoo | 22 yrs/M | U | Student | 72(SBP) | - | 112.00 | 2.20 | 110.0 | 3.2 | 8.20 | 4.00 | 6.20 | N | Internal Haemorrhage | A | WNL | 13.4 | E |
| 21. | Jitendra Singh | 58 yrs/M | M | Service | 60(SBP) | Anuria | 98.00 | 2.40 | 148.0 | 4.2 | 8.80 | 3.00 | 5.90 | N | Cardiogenic shock with MI | A | Nil | 10.6 | E |
| 22. | Asha | 26 yrs/F | M | House wife | 90/70 | - | 128.00 | 2.00 | 118.0 | 4.8 | 10.60 | 4.60 | 3.40 | N | AGE | A | Nil | 13.6 | D1 |
| 23. | Jharkote | 68 yrs/m | M | Farmer | 118/70 | - | 155.00 | 4.30 | 148.0 | 4.2 | 9.20 | 5.60 | 4.40 | E | BPH | C | Alb+, acc. Pus cell + | 12.8 | D2 |
| 24. | Tulsidas | 60 yrs/M | M | Business | 80(SBP) | ++ | 120.00 | 2.00 | 144.0 | 4.2 | 10.50 | 6.80 | 4.60 | N | AGE | A | Nil | 7.2 | D1 |
| 25. | Dam Khan | 20 yrs/M | U | Student | 132/80 | + | 84.00 | 2.00 | 142.0 | 4.6 | 10.50 | 3.20 | 5..70 | N | Malaria | B | Alb-traces | 10.2 | G |

# MASTER CHART

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26. | Sunita | 28 yrs/F | M | House wife | 76(SBP) | ++ | 84.00 | 3.80 | 122.0 | 5.6 | 9.80 | 7.40 | 5.20 | N | Burn | A | Nil | 16.2 | E |
| 27. | Rajaram | 68 yrs/M | M | Farmer | 156/90 | - | 94.00 | 1.60 | 108.0 | 6.5 | 7.60 | 6.20 | 3.40 | N | AGE | A | Nil | 16.2 | D1 |
| 28. | Shyam | 18 yrs/M | U | Student | 46(SBP) | +++ | 146.00 | 3.80 | 138.0 | 4.2 | 10.80 | 5.00 | 4.60 | N | Celphas poison | B | Nil | 10.6 | E |
| 29. | Nandu | 40 yrs/M | M | Business | 110/76 | + | 106.00 | 1.60 | 138.0 | 4.2 | 8.80 | 3.20 | 6.50 | N | Malaria | B | Alb +++ | 13.0 | D1 |
| 30. | Panka | 20 yrs/M | U | House wife | 138.80 | + | 146.00 | 3.30 | 126.0 | 5.7 | 10.20 | 3.80 | 6.90 | N | Hepatic coma | B | Nil | 11.5 | E |
| 31. | Kankaiya Lal | 72 yrs/M | M | Farmer | NR | Anuria | 212.00 | 3.60 | 108.0 | 3.4 | 9.50 | 2.80 | 5.80 | N | AGE | A | Nil | 14.2 | D1 |
| 32. | Sushila | 22 yrs/F | M | House wife | 76(SBP) | ++ | 100.00 | 2.80 | 132.0 | 6.2 | 10.40 | 2.80 | 6.50 | N | Burn | A | Nil | 12.6 | E |
| 33. | Shiv Pal Singh | 68 yrs/M | M | Service | 120/72 | + | 98.00 | 1.80 | 138.0 | 5.6 | 9.30 | 4.20 | 5.20 | N | Diabetes | A | Alb + | 10.8 | D2 |
| 34. | Saroj | 32 yrs/M | M | House wife | 96/62 | + | 168.00 | 2.40 | 136.0 | 5.2 | 9.80 | 3.20 | 6.90 | N | Septicemia | B | Acc. Puss cell + | 7.8 | E |
| 35. | Narayan | 36 yrs/M | M | Business | 90/70 | + | 108.00 | 2.30 | 132.0 | 4.8 | 10.30 | 4.90 | 7.10 | N | AGE | A | Nil | 10.6 | F |
| 36. | Sushil | 12 yrs/M | U | Student | 98/64 | - | 109.00 | 2.70 | 136.0 | 5.2 | 10.80 | 2.70 | 3.40 | N | AGE | A | Nil | 10.3 | D1 |
| 37. | Rameshwar | 32 yrs/M | M | Service | 80/42 | Anuria | 122.00 | 1.90 | 108.0 | 4.6 | 10.00 | 3.10 | 5.50 | N | Blood loss Anemia | A | Nil | 13.4 | E |
| 38. | Ramkali | 40 yrs/F | M | House wife | NR | Anuria | 71.00 | 2.30 | 127.0 | 5.1 | 8.80 | 3.10 | 6.10 | N | Hypo volumic shock | A | Nil | 10.8 | D2 |
| 39. | Munna Lal | 36 yrs/M | M | Worker | 82/50 | - | 149.00 | 1.80 | 117.0 | 4.6 | 9.60 | 4.90 | 6.70 | N | Cirrhosis with PHT | A | Nil | 10.7 | D2 |
| 40. | Mulli | 76 yrs/M | M | Farmer | 100/62 | + | 198.00 | 4.20 | 138.0 | 4.6 | 11.00 | 5.30 | 6.80 | N | AGE | A | Nil | 10.8 | D1 |
| 41. | Savitri | 38 ytrs/F | M | House wife | 80/60 | + | 128.00 | 2.30 | 136.0 | 3.8 | 9.60 | 4.10 | 3.60 | N | CHF with RHD, MS, MR | A | Nil | 11.2 | D2 |
| 42. | Ram Shree | 54 yrs/F | M | House wife | 106/70 | + | 90.00 | 2.60 | 136.0 | 6.8 | 9.70 | 4.30 | 4.40 | N | AGE | A | Nil | 9.6 | G |
| 43. | Prem Bai | 36 yrs/F | M | House wife | 94/60 | - | 86.00 | 2.10 | 138.0 | 5.0 | 8.20 | 4.30 | 6.80 | N | Pericardial effusion | A | Nil | 10.6 | D1 |
| 44. | Rani | 20 yrs/F | U | Student | 102/70 | - | 106.00 | 2.00 | 138.0 | 5.5 | 10.60 | 3.40 | 4.60 | N | AGE | A | Nil | 9.8 | D1 |
| 45. | Kunti | 28 yrs/F | M | House wife | 68(SBP) | Anuria | 104.00 | 2.40 | 136.0 | 4.7 | 6.80 | 5.30 | 5.80 | N | PPH | A | Nil | 12.3 | E |
| 46. | Suraj Prasad | 43 yrs/M | M | Business | 100/64 | + | 88.00 | 1.80 | 132.0 | 5.2 | 9.20 | 3.50 | 5.40 | N | AGE | A | Nil | 10.3 | D1 |
| 47. | P.D. Shukla | 43yrs/M | M | Service | 110/70 | - | 96.00 | 1.60 | 137.0 | 3.6 | 9.20 | 3.90 | 2.80 | N | Food poisoning & diarrhoea | A | Nil | 10.6 | D1 |
| 48. | Anil Kumar | 28 yrs/M | U | Student | 82(SBP) | Anuria | 104.00 | 1.90 | 143.0 | 5.6 | 8.30 | 4.20 | 3.80 | N | Accident with shock | A | Nil | 8.3 | E |
| 49. | Suman | 22 yrs/F | M | House wife | 68(SBP) | Anuria | 82.00 | 1.80 | 113.0 | 7.6 | 9.00 | 3.70 | 6.20 | N | Burn | A | Nil | 16.3 | E |
| 50. | Suresh | 28 yrs/M | M | Service | 114/70 | + | 78.00 | 2.00 | 126.0 | 5.3 | 9.40 | 4.90 | 3.20 | N | AGE | A | Nil | 11.7 | D1 |

M= Married U= Unmarried E= Expired D= Discharged A= Pre renal B= Renal C= Post renal D1= Discharge after complete recovery D2= Discharge after partial recovery E= Expired F= Absconded G= LAMA